at the same, and at all times, integrating their members into the policies and aims of the general organization.

* * *

Wherein Nonmetropolitan Societies Are Fortunate.—The members of nonmetropolitan societies may congratulate themselves upon their good fortune in being able to know one another with considerable intimacy and, through their organization, to derive not only pleasure from the meetings, but profit in a considerable degree from association with one another, as they daily go about in the practice of their profession. It is gratifying to note that most of the smaller county medical societies appreciate the possible benefit through a preliminary dinner, or a postmeeting supper, as part of the regular procedure. The value of such social sessions, indeed, cannot be overestimated. We cannot break bread with and also dislike or hate one another.

* * *

Concerning Scientific Programs.—A few comments concerning the scientific papers. The program committee (of whoever it may be composed: the secretary, the president, or a special group) should map out, if possible, its program in advance. Such a plan is satisfactorily carried through in many societies, and with some effort it can be made practical in all organizations. Do not depend altogether on outside speakers. The members of a medical society profit most in proportion as they themselves make an effort to contribute papers on medical problems coming under their immediate observation. This is particularly the case if such presentations are followed by free and kindly discussion participated in by other local colleagues. The out-of-county speaker should be on the occasional, rather than on the regular program.

To stimulate extra interest, clinic days may be arranged, to occur from time to time. The State Association is prepared to aid in this, and the Association Secretary will coöperate in all such endeavors.

* * *

Civic Obligations.—Let it not be forgotten, too, that in November of this year, less than a month away, the State election will be held. At that time the State assemblymen and State senators who make up the legislature will be elected.

The members of every county society should know the attitude on public health and medical practice questions of all such legislative candidates. "An ounce of prevention is worth a pound of cure." Find out what you want to know in advance, not afterward: for no candidates who are opposed to legitimate public health or medical practice standards should be supported by physicians. More than that, every proper and diplomatic method should be used to prevent the election of such candidates.

The 1937 California Legislature will have before it many items of grave importance to medical men and women. One of these, the codified Medical Practice Act, is discussed editorially in this same issue of CALIFORNIA AND WESTERN MEDICINE. Officers of county societies who fail to call the attention of their members, before the legislature assembles, to the importance of civic obligations may have reason to reproach themselves after the legislature adjourns!

* * *

To Summarize.—On our immediate needs, which are: stimulating professional programs, best possible set-ups for fraternal and social understandings, and alertness to civic responsibilities in relation to public health and medical practice standards. Those should be some of our major objectives in the present and immediate future.

Other State Association and Component County Society News.—Additional news concerning the activities and work of the California Medical Association and its component county medical societies is printed in this issue, commencing on page 364.

EDITORIAL COMMENT†

THE PULSE PRESSURE—PULSE RATE EXERCISE TOLERANCE TEST

Since 1889 the effects of exercise on the blood pressure and pulse rate have been studied and used as tests of efficiency of the circulatory system. The difficulty in drawing conclusions from various reactions has been that individuals with effort syndrome, but no organic heart disease—the so-called "irritable hearts" and "soldiers' hearts," showed extreme fluctuations of blood pressure and pulse rate. Most tests gave as the normal response a rise of the systolic blood pressure and pulse rate to a given degree, *i. e.,* blood pressure 120 to 140 systolic, and pulse of from 80 to 100 with a fixed exercise. A normal reaction should show a return to previous levels within two and one-half minutes. Often formulae were developed, such as in the Schneider test. T. R. Harrison estimated cardiac efficiency by measuring dyspnea after fixed exercise, developing the formula for a normal ventilation quotient.

Master devised as an exercise tolerance test the ability of the blood pressure and pulse rate to return to within ten points of normal levels within two minutes after cessation of exercise. He wisely presented tables of normal degree of exercise for different ages and weights (Table 1). The figures represent the number of ascents of a 1½-foot, 2-step climb in one and one-half minutes, which, if multiplied by weight, equal foot pounds per minute of work (excluding work of descent). Some

† This department of CALIFORNIA AND WESTERN MEDICINE presents editorial comment by contributing members on items of medical progress, science and practice, and on topics from recent medical books or journals. An invitation is extended to all members of the California Medical Association to submit brief editorial discussions suitable for publication in this department. No presentation should be over five hundred words in length.

TABLE 1

A. Standard Number of Ascents for Males

Weight (lb.)	Age in Years 5–9	10–14	15–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69
40–49	35	36	....	....	....	....	....	....	....	....	....	....	....
50–59	33	35	32	....	....	....	....	....	....	....	....	....	....
60–69					....	....	....	....	....	....	....	....	....
70–79	28	32	30	....	....	....	....	....	....	....	....	....	....
80–89	26	30	29	29	29	28	27	27	26	25	25	24	23
90–99	24	29	28	28	28	27	27	26	25	25	24	23	22
100–109	22	27	27	28	28	27	26	25	25	24	23	22	22
110–119	20	26	26	27	27	26	25	25	24	23	23	22	21
120–129	18	24	25	26	27	26	25	24	23	23	22	21	20
130–139	16	23	24	25	26	25	24	23	23	22	21	20	20
140–149	....	21	23	24	25	24	24	23	22	21	20	20	19
150–159	....	20	22	24	25	24	23	22	21	20	20	19	18
160–169	....	18	21	23	24	23	22	22	21	20	19	18	18
170–179	....	....	20	22	23	23	22	21	20	19	18	18	17
180–189	....	....	19	21	23	22	21	20	19	19	18	17	16
190–199	....	....	18	20	22	21	21	20	19	18	17	16	15
200–209	....	....	....	19	21	21	20	19	18	17	16	16	15
210–219	....	....	....	18	21	20	19	18	17	17	16	15	14
220–229	....	....	....	17	20	20	19	18	17	16	15	14	13

B. Standard Number of Ascents for Females

Weight (lb.)	Age in Years 5–9	10–14	15–19	20–24	25–29	30–34	35–49	40–44	45–49	50–54	55–59	60–64	65–69
40–49	35	35	33	....	....	....	....	....	....	....	....	....	....
50–59	33	33	32	....	....	....	....	....	....	....	....	....	....
60–69	31	32	30	....	....	....	....	....	....	....	....	....	....
70–79	28	30	29	....	....	....	....	....	....	....	....	....	....
80–89	26	28	28	28	28	27	26	24	23	22	21	21	20
90–99	24	27	26	27	27	26	25	24	23	22	21	20	19
100–109	22	25	25	26	26	25	24	23	22	21	20	19	18
110–119	20	23	23	25	25	24	23	22	21	20	19	18	18
120–129	18	22	22	24	24	23	22	21	20	19	19	18	17
130–139	16	20	20	23	23	22	21	20	19	19	18	17	16
140–149	....	18	19	22	22	21	20	19	19	18	17	16	16
150–159	....	17	17	21	20	20	19	19	18	17	16	16	15
160–169	....	15	16	20	19	19	18	18	17	16	16	15	14
170–179	....	13	14	19	18	18	17	17	16	16	15	14	13
180–189	....	....	13	18	17	17	17	16	16	15	14	14	13
190–199	....	....	12	17	16	16	16	15	15	14	13	13	12
200–209	....	....	....	16	15	15	15	14	14	13	13	12	11
210–219	....	....	....	15	14	14	14	13	13	13	12	11	11
220–229	....	....	....	14	13	13	13	13	12	12	11	11	10

dyspnea results, which represents an oxygen debt. This test alone is of much value, but in recent years the author has used a test of heart function depending on the general assumption that widening-pulse pressure is an index of increased systolic heart output. Starr, Y. Henderson, Bock, Burwell and others have shown that there is a correlation between a rising-pulse pressure and an increased systolic heart output, and Ogden and Shock have worked with normal adolescents (an unfortunately unstable group) in attempting to demonstrate this correlation. Bazett and his collaborators recently have proved this relationship, using a special technique for blood pressure recording.

It is well known that in heart failure the pulse pressure narrows and the pulse rate rises, improvement often being measured by reversal of this state. The logical reason for this is that the minute output of the heart of oxygenated blood for the tissues must be maintained by a rising pulse rate (through the Bainbridge reflex) when the output per beat falls. Exercise to the point of dyspnea puts an increased demand on any heart by producing an oxygen debt. This should roughly tell us whether the muscle is able, through diastolic dilatation and forceful contraction, as represented by the pulse pressure, to proportionately take care of the increased minute output, or if the increased rate must account for most of it. One can combine the Master's test of variable amounts of exercise, using a one-foot step for one minute, with an analysis of the proportion of increase of pulse pressure and pulse rate to tell how the heart is taking care of the minute output demanded. The normal may show figures as follows: Resting blood pressure 120/80 = 40 pulse pressure, 60 pulse rate; after exercise, blood pressure 140/80 = 60 pulse pressure (a 50 per cent increase) and 90 pulse rate (a 50 per cent increase). A patient with myocardial damage, on the contrary, characteristically shows figures such as: Resting blood pressure 120/80 = 40 pulse pressure, 60 pulse rate; after exercise, blood pressure 130/80 = 50 pulse pressure (a 25 per cent increase) and 120 pulse rate (a 100 per cent increase). Such a patient, after his Standard Master's exercise, would not return to a normal condition in blood pressure and pulse rate in from two to two and one-half minutes.

Patients with complete heart-block illustrate this phenomenon particularly well. Since their pulse rate is fixed, their only means of increasing minute cardiac output is by increased systolic output per beat, *i. e.*, resting blood pressure 160/80, pulse rate 40; after exercise, blood pressure 240/80 = 100 per cent increase, pulse rate 40. Occasionally, the duration of systolic output is prolonged, *i. e.*, aortic stenosis, which would destroy the correlation of pulse pressure increase and systolic output increase. Naturally, aortic insufficiency would distort the estimates, and marked arteriosclerosis of the great vessels tend to exaggerate the pulse pressure reactions. In patients

with angina pectoris, occasionally the pulse rate does not rise proportionately to pulse pressure. Even in unstable individuals, *i. e.*, adolescents and patients showing effort syndrome, it is rare that a marked disproportion is encountered even though both pulse and blood pressure responses are exaggerated and remain abnormally elevated.

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CARDIAC MURMURS

The stethoscope is of little clinical value unless the physician understands the significance of the sounds he hears. When the auscultatory method is properly applied to the circulatory system, the ability to diagnose certain functional disturbances is greatly enhanced, and the nature of lesions predicated. Fortified by this evidence, prognosis and treatment may be more rationally directed.

Cardiac murmurs may arise from several sources; some external to the heart, but all associated in some manner with its contraction. In growing children and young adults, faults in stream-lining of the heart and great vessels are probably responsible for most of the so-called functional murmurs. They are usually systolic in time and localized. Cardiorespiratory murmurs are also common at this age and have no clinical significance. Pericardial or pleuropericardial friction sounds are also differentiated from murmurs of valvular origin.

Murmurs arising in conjunction with valvular defects are important. Their location in the chest, direction of transmission, intensity, pitch and time in the cardiac cycle determine their point of origin. The effects of exercise and change in posture may aid in interpretation.

In studying the propagation of cardiac murmurs, it is well to remember that they travel on or with the blood current in somewhat the same fashion as audible sound travels on the wind. It is easier to whistle down the wind than up the wind.

Variations from the normal cardiac sounds and murmurs in the periphery will be taken up at another time.

Systolic murmurs at the base tend to be rough; if from the aortic valve, to be transmitted upward along the course of the larger branches of the aorta, and if from the pulmonic valve, to be transmitted more readily toward the left clavicle. They are frequently accompanied by a systolic thrill. If the aortic valve is more than slightly stenosed, the pulse is of the plateau type. A subject with a dilated aorta or dilated arteries arising from the arch may present a systolic murmur and thrill over the upper sternum and vessels at the root of the neck. This must be differentiated from aortic stenosis.

Aortic diastolic murmurs are usually soft, tend to replace the second aortic sound and are transmitted toward the apex, often fading over the mid-cardiac region where the right ventricle overlies the left ventricle, and becoming more audible again at the apex. A mid-diastolic rumble accompanied by a thrill at the apex may be noted in a few cases, and in a considerable number of cases a presystolic murmur (the Austin Flint murmur) and thrill will be observed at the apex. (The driving character of the aortic diastolic murmur is notable.)

Pulmonary diastolic murmurs (Graham Steele type) are usually of brief duration, localized to the second and third left interspaces close to the sternum. They are usually associated with right cardiac failure and are, therefore, generally transient. We have described a new murmur, presystolic in time, heard best at the tricuspid region in pulmonary insufficiency, and probably having the same mechanism as the Austin Flint murmur in aortic insufficiency. This murmur is also heard in right-sided failure and is transient.

Mitral systolic murmurs are heard best at the apex and are transmitted to the left and back. They are high-pitched and blowing in character. The harsh rasping systolic murmur at the apex in cardiosclerosis is most frequently associated with hypertrophy and dilatation of the heart, and sclerosis of the mitral valve. Exercise generally increases the intensity of the murmurs of mitral insufficiency.

Mitral diastolic murmurs may be early, middle, or late (presystolic). The auricular or presystolic murmur disappears with the onset of auricular fibrillation in the absence of aortic insufficiency. In rapid hearts the various phases of the murmur cannot be accurately recognized as the rumbling presystolic crescendo and loud first sound predominate, especially after exercise.

Tricuspid systolic murmurs are almost the rule in congestive heart failure. They are heard best over a small area at the lower sternum, just to the left of the lower sternum or in the epigastrium. They are of brief duration, "close to the ear," and often have an amphoric quality. With organic tricuspid valvulitis the murmur may be very harsh, and accompanied by a thrill.

Tricuspid diastolic murmurs are relatively uncommon and are usually associated with tricuspid stenosis. They may be early or late in diastole (presystolic). The presystolic phase disappears with the onset of auricular fibrillation.

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CHRONIC VITAMIN D TOXICITY

A warning against the possibility of acute or chronic poisoning from excessive administration of vitamin D preparations was recently made in these columns.[1] It was pointed out that there is as yet no evidence to indicate what the chronic toxicity of vitamin D preparations may be, especially if contaminated with "toxisterol." This poisonous ingredient, which may be present in any irradiated ergosterol product if the irradiation is

1 Leake, C. D.: Calif. and West. Med., 44:149 (March), 1936.